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SHORT REPORT

High Output Cardiac Failure Caused by Popliteal Pseudoaneurysm and Arteriovenous Fistula Following Total Knee Replacement Simulating Severe Aortic Stenosis

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Introduction

Popliteal pseudoaneurysm formation is a rare complication following total knee replacement (TKR).[1] The development of arteriovenous fistula (AVF) is a rare but recognised complication of any form of aneurysmal disease of the popliteal artery and leads to an increase in cardiac output.[2] The development of high output cardiac failure as a consequence of AVF formation is uncommon but well recognised.

Aortic valve disease is relatively common and is a well recognised cause of cardiac failure. The combination of severe aortic stenosis and consequent cardiac failure is usually an indication for aortic valve replacement.

We describe one case in which high output cardiac failure due to the formation of a popliteal pseudoaneurysm and subsequent development of an AVF as a complication of TKR simulated severe aortic stenosis. We also illustrate the ultrasonographic and angiographic findings.

Case Report

A 61 year old diabetic, retired computer operator underwent a right TKR for osteoarthritis. Six weeks following the surgery he presented to the Accident and Emergency department with sudden, severe shortness of breath, atrial fibrillation and an ejection systolic murmur. His chest X-ray demonstrated pulmonary oedema and appropriate therapy was commenced, leading to resolution of his cardiac symptoms.

A transthoracic echocardiogram was performed and revealed an instantaneous aortic valve gradient of 65 mmHg and good left ventricular function. As no other cause for his heart failure could be identified, he was referred for consideration for aortic valve replacement and, as part of his work up, underwent coronary angiography. This showed non-critical coronary artery disease and a peak to peak aortic valve gradient of 30 mmHg. Both the referring cardiologist and the cardiac surgeon, while accepting that the patient had haemodynamically significant aortic valve disease, commented that its apparent clinical severity was greater than would be expected based on the echocardiographic appearance. For this reason, and because the patient's symptoms were well controlled, valve replacement was deferred.

Two months later he presented with right foot ulceration, ischaemic rest pain in his right leg and breathlessness on minimal exertion equivalent to New York Heart Association (NYHA) class 3. His right leg was swollen and a pulsatile mass was palpable behind the right knee. Ultrasound duplex imaging showed a popliteal aneurysm with an AVF.

Fig. 1(a)–(c) shows the popliteal aneurysm and AVF with high velocity, pulsatile flow through the popliteal artery and vein.

Peripheral angiography was subsequently performed.

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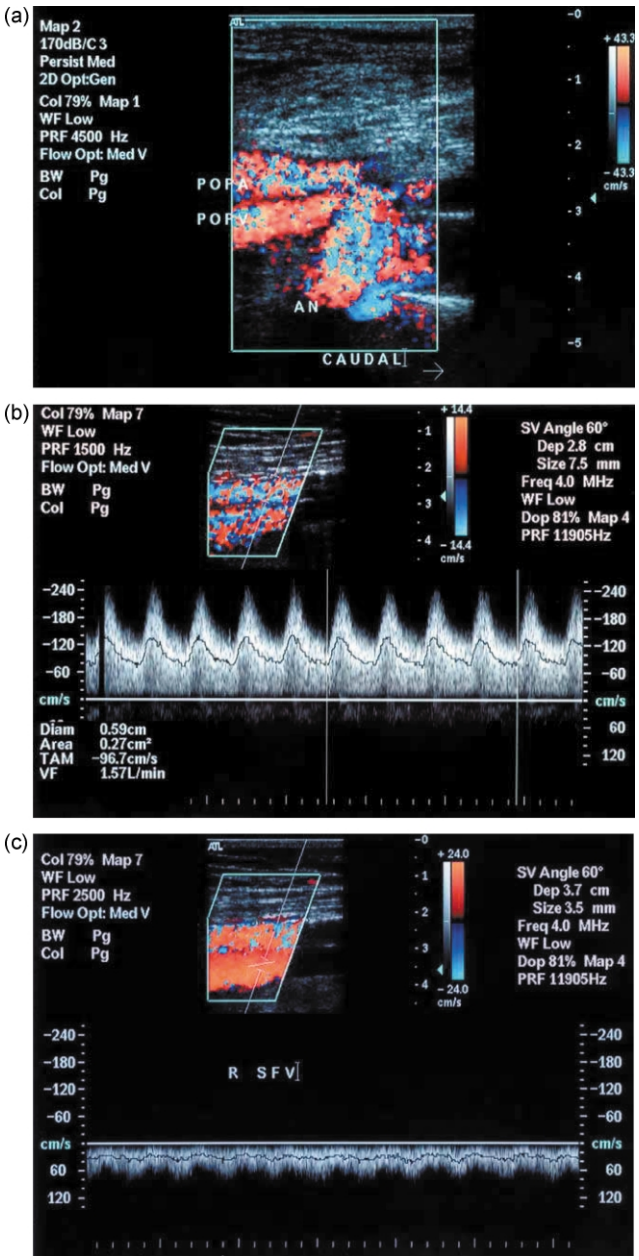

Fig. 1. Ultrasound duplex scan. (a) The popliteal pseudoaneurysm and AVF. (b) Shows high velocity, pulsatile flow in the superficial femoral artery. (c) Shows high velocity, pulsatile flow in the superficial femoral vein.

Fig. 2(a)–(c) shows the popliteal aneurysm with early filling of the popliteal vein through a large AVF and poor distal run off via anterior tibial artery.

The patient underwent successful surgical repair of the popliteal AVF. The popliteal AVF was approached via a lazy 'S' incision. The popliteal artery and then the vein proximal and distal to the fistula were controlled. The fistula and associated pseudoaneurysm were opened and the popliteal vein repaired by lateral suture. The pseudoaneurysm was resected and the popliteal artery repaired with an interposition long saphenous vein graft harvested from the contralateral groin.

Post-operatively his leg pain and breathlessness improved (NYHA Class 1), as did his general health. His cardiac failure medications are currently being reduced. A post-operative transthoracic echocardiogram revealed an instantaneous gradient of 50 mmHg.

Discussion

A recent review of the literature found that local vascular complications following TKR are rare, occurring between 0.03 and 0.2% of cases.[3]

The least uncommon such complications are arterial thrombosis, arterial transection, direct AVF and aneurysm formation. Only 1 out of 60 reported cases (1.8%) developed an AVF of the geniculate vessels.[3] In one study such complications were confined to those patients with pre-existing peripheral vascular disease (PVD).[4] So possibly, the presence of risk factors for PVD, such as smoking, diabetes, hypertension, hypercholesterolaemia and a strong family history, and also those with claudication or rest pain, should prompt consideration of vascular consultation prior to knee replacement.[1]

Local vascular complications have been reported to present in a number of ways including acute leg ischaemia, haemorrhage as a result of direct vessel trauma, swelling due to aneurysm or AVF formation and recurrent haemarthroses. To the best of our knowledge, our report is the first to describe presentation with dyspnoea and pulmonary oedema.

There are several causes of AVF. Congenital causes include Klippel–Trénaunay syndrome and Sturge–Weber syndrome. Acquired causes include atherosclerotic degeneration, aneurysm formation and rupture of an aneurysm into the adjacent vein, iatrogenic damage, mycotic aneurysm formation (infection weakening the arterial wall) with subsequent rupture into the adjacent vein, neoplastic erosion and penetrating trauma, e.g. from gunshot or stab wounds.

Popliteal pseudoaneurysm formation

Aneurysm formation and fistulation usually present as delayed complications 4–6 weeks post-operatively. The commonest causes, though extremely rare, are due to direct injury to the popliteal or geniculate vessels. This can occur due to direct laceration, blunt damage during vessel retraction and secondary to

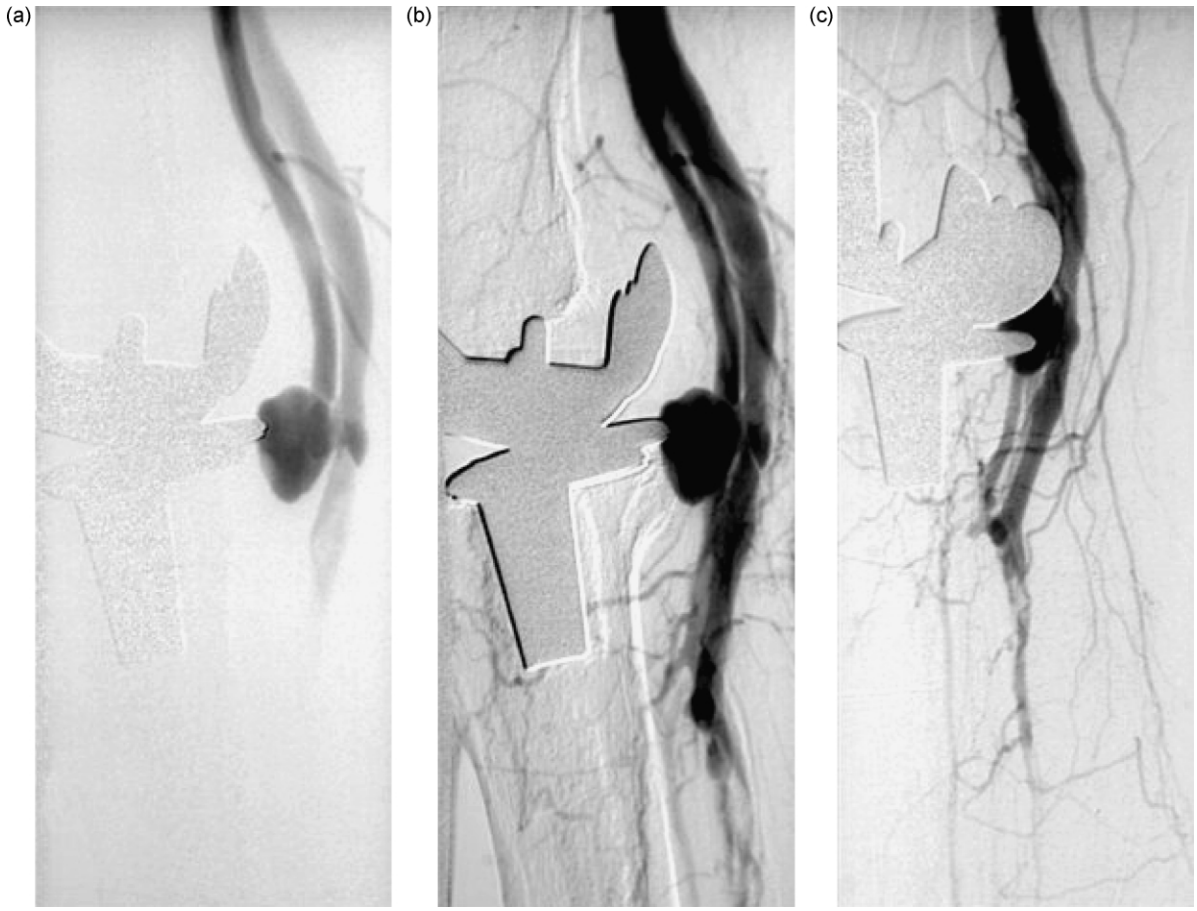

Fig. 2. Digital subtraction angiography. (a) The popliteal pseudoaneurysm and AVF with early filling of the popliteal vein. (b) Continued opacification of the popliteal pseudoaneurysm and AVF behind the total knee arthroplasty. (c) Subsequent image showing poor distal run off via the anterior tibial artery.

penetrating injury from surgical fixation screws. In the knee, post-operative geniculate artery aneurysms are far commoner than popliteal artery aneurysms.[5]

The initial event is haematoma formation in the tissue surrounding the damaged artery. This then organises with fibrinisation and connective tissue formation, followed by endothelialisation of a central cavity. If this cavity communicates with the damaged popliteal vessel a pseudoaneurysm is created which may then fistulate into an adjacent vein due to inflammatory necrosis of the vessel walls thus forming an AVF.

Definitive diagnosis of AVF is usually confirmed by non-invasive duplex ultrasonography or arteriography.

The treatment options for AVF may be conservative, surgical or endovascular. Some will resolve spontaneously over 3–6 months. If spontaneous resolution does not occur or if symptoms dictate then surgical repair may be indicated. Good results are achieved by excision and vascular reconstruction. Endovascular repair is possible in appropriately selected cases depending on aneurysm and AVF size, risk of peripheral embolization and systemic symptoms. Techniques used include catheter-delivered occlusion coils, balloon occlusion and stenting across the fistula.[6]

In our patient, surgical repair was chosen as the fistula was too large for endovascular closure.

High output cardiac failure and aortic valve disease

A large arteriovenous shunt can lead to a significantly increased cardiac output and may precipitate cardiac failure even in an otherwise normal heart. High output cardiac failure due to AVF has been reported with dialysis access fistulas, post-nephrectomy renal AVF or aortocaval fistulae, intracranial AVFs and with intrahepatic and/or intrapulmonary lesions in patients with hereditary haemorrhagic telangiectasia. There has been one reported case of high output cardiac failure and simulated prosthetic aortic valve

dysfunction as a result of a femoral vessel AVF which developed 14 years after femoral artery puncture at the time of cardiac catheterisation.[7] High output cardiac failure precipitated by a popliteal AVF has not previously been reported.

Severe aortic stenosis is a recognised cause of congestive cardiac failure and typically presents acutely with a low volume pulse, an ejection systolic murmur and pulmonary oedema.

The severity of aortic valve stenosis can be calculated by measuring the velocity of the blood ejected from the left ventricle through the aortic valve using Doppler echocardiography. Echocardiography gives more information about the valve and left ventricular function than cardiac catheterization but cannot provide the information on coronary arterial anatomy that is required if valve replacement is being considered.

In the presence of a large arteriovenous shunt, the gradient across a stenotic aortic valve is increased and the severity of the stenosis may be overestimated. Our patient had a tricuspid aortic valve with a preoperative maximal peak instantaneous Doppler gradient of 65 mmHg and good left ventricular function on transthoracic echocardiography. A post-operative study demonstrated a decrease in peak instantaneous gradient to 50 mmHg. It has been shown in several animal studies that AVF created surgically lead to an immediate increase in heart rate, cardiac output, stroke volume, left and right atrial and pulmonary artery pressures. There is also a concomitant decrease in mean arterial pressure, effective systemic blood flow and pulmonary and systemic vascular resistance.

In adults, aortic valve replacement is recommended once aortic stenosis becomes symptomatic. Even in asymptomatic patients, some authorities would consider replacement of valves with a gradient >50 mmHg. If a potentially reversible cause of cardiac failure existed other than aortic stenosis, which if corrected led to resolution of symptoms, then most authorities would not consider valve surgery indicated.

The mean survival is only 1–2 years in patients who develop symptoms of heart failure due to aortic stenosis and this condition results in low output cardiac failure.

Differentiation between high and low output cardiac failure is not always easy. Invasive haemodynamic measurements are the gold standard for diagnosis. However, these are not always indicated in the presence of what otherwise appears to be a clear-cut clinical picture.

If high output cardiac failure is diagnosed, there should then follow a systematic workup to exclude the principal causes which include AVF, anaemia, hyperthyroidism, myeloproliferative disorders, Paget's disease, pregnancy, sepsis and thiamine deficiency.

In our patient, pulmonary oedema was probably precipitated by a combination of increased cardiac output, due to shunting of blood through the newly formed AVF, and pre-existing aortic stenosis of which subsequently proved to be of moderate severity. Since the contribution of the AVF to the patient's condition was not initially recognised, the severity of the aortic stenosis was overestimated.

Conclusion

1. Popliteal AVF formation following TKR is rare.
2. The clinician should remain aware of the adverse haemodynamic effects of AVF.
3. Careful preoperative evaluation is necessary prior to knee replacement. If risk factors for, or symptoms of PVD are present then a vascular consultation should be considered. These patients may also have undiagnosed coronary/valvular heart disease.
4. Vascular referral should be prompt post-TKR if acute vascular complications are suspected and particular care should be taken in patients with valvular heart disease and/or cardiac failure.
5. In patients presenting with new onset cardiac failure post-TKR, the possibility that a high cardiac output secondary to AVF is the cause or is a contributing factor should be considered.
6. A potentially reversible cause of high output cardiac failure is rarely present.

Acknowledgements

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